Lipid composition of parenteral nutrition acutely affects hepatic lipid deposition and lipid metabolism in preterm pigs

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experimental colitis+ spiriluna group when compared with experimental colitis group ($p < 0.05$). Immunoreactivity was seen to be decreased in experimental colitis+ spiriluna group when compared with experimental colitis group ($p < 0.05$). Tissue MDA levels was seen to be decreased in Experimental colitis+ Spirulina group when compared with Experimental colitis groups ($p < 0.05$).

Conclusion: The present study indicates the beneficial efficacy of spirulina extract in TNBS-induced inflammatory bowel disease.

Disclosure of Interest: None declared

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DIETARY ALPHA-LINOLENIC ACID-RICH FORMULA REDUCES THE EXPRESSION OF ICAM-1 AND VCAM-1 IN RATS WITH TNBS-INDUCED COLITIS INDEPENDENTLY OF HO-1

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Rationale: We have previously shown that a-linolenic acid inhibits pro-inflammatory production of interleukin-8 in enterocyte-like Caco-2 cells in response to a pro-inflammatory stimulus [1]. We then demonstrated that dietary a-linolenic acid-rich oil reduces inflammation and oxidative stress in an animal model of colitis [2]. We now aimed to evaluate whether dietary a-linolenic acid may reduce the expression of adhesion molecules by inducing the protective enzyme heme-oxygenase-1 in a colitis model.

Methods: Colitis was induced at d0 by intrarectal injection of 2–4-6-trinitrobenzen sulfonic acid (TNBS) whereas control rats received the vehicle. Rats received either 450 mg/kg/d of ALA, or an isocaloric corn oil formula (control and TNBS groups) for 14 d (from d –7 to d 7). The colonic expression of ICAM-1, VCAM-1 and HO-1 was studied by immunohistochemistry using specific antibodies.

Results: TNBS challenge significantly increased ICAM-1, VCAM-1 and HO-1 expression compared to the control group (both $p < 0.001$). ALA-rich diet significantly decreased the expression of ICAM-1 and VCAM-1 compared to the TNBS group (both $p < 0.001$), but it did not affect the expression of HO-1.

Conclusion: These findings show that ALA-rich formula decreases the expression of ICAM-1 and VCAM-1 independently of HO-1. Modulation of the endothelial response may contribute to the anti-inflammatory effects of ALA during intestinal inflammation.

References

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[2] Hassan A et al., J Nutr, in press.

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PP270

USEFUL ADMINISTRATION OF LIPID MIXTURE TO LIVER RESECTED AND PANCREATITIS PATIENTSE

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Rationale: Administration of lipid mixture to liver resected patients and patients affected by severe pancreatitis (SP), should be useful to supply high amount of calories on small fluid quantity, moreover it can reduce hyper-inflammatory reaction by omega-3 fatty acids supply.

Methods: Medium-chain (MCT) and long-chain fatty acids (with the latter including, on total fatty acids, saturated fatty acids 16:0 and 10:0 11.9%, omega-3 polyunsaturated fatty acids (PUFA) 18:3, 20:4 and 22:6 7.0%, omega-6 PUFA 18:2 and 20:4 19.2% and omega-9 monounsaturated fatty acids 18:1 27.8% mix by parenteral way plus carbohydrates, aminoacids and fluid) to a patient treated by extended right liver resection and to 3 patients by SP. All these 3 patients have been supplied by enteral nutrition too. Parenteral mixture supplied 100 g of amino acids (~1 g/kg), 1800 kcal (~19 kcal/kg) divided between 70% carbohydrates as dextrose solution and 30% fats as lipid emulsion based on MCT 30%, soybean oil 30%, olive oil 25%, and fish oil 15%, besides micronutrients and electrolytes. Definitive pathologic examination stated cholangiocarcinoma in cirrhotic liver after a primitive diagnosis of steatosis, while diagnosis of infected pancreatitis has been confirmed at surgery.

Results: Postoperative course of PS patients was complicated by colonic leakage (1), pancreatic collection infection (1), treated by endoscopic cleaning (2), biliary leakage treated by percutaneous biliary drainage (1) and gastric suction tube. CRP decreased during first 2 weeks from 315 to 45 mg/L. All the patients have been discharged and well being.

Conclusion: Cirrhotic patients are at metabolic risk after liver resection, while patients with severe pancreatitis at infectious risk. Anyway, PUFA and lipids could be useful to reduce impact of inflammatory cascade and decreases reaction in peritoneal tissue as well as on cirrhotic liver.

Disclosure of Interest: None declared

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Outstanding abstract

LIPID COMPOSITION OF PARENTERAL NUTRITION ACUTELY AFFECTS HEPATIC LIPID DEPOSITION AND LIPID METABOLISM IN PRETERM PIGS

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Rationale: Total parenteral nutrition (TPN) can be life-saving for patients who do not tolerate enteral nutrition. However the optimal composition of TPN and effects of partial enteral nutrition (PEN) remain unclear.

Methods: 78 preterm pigs were delivered by caesarean section at 92% of gestation and assigned to isoenergetic TPN groups. Varying ratios of n-6:n-3 polyunsaturated fatty acids (PUFA) were fed to four groups using a standard TPN (S: Nutriflex Lipid Plus, Braun, n-6:n-3

PUFA-ratio of 7:1): no addition (S, n = 19), addition of fish oil emulsion (33% of total lipids; S+O, n =19, Omegaven/Fresenius Kabi, final n-6:n-3 PUFA-ratio of 1.5:1), replacement of lipids with dextrose (NL, n = 22) and enteral colostrum to the standard (~60% of energy; S+PEN, n = 18). After 3 days of differential feeding, all pigs were shifted to 2 days of total enteral nutrition with infant formula. Plasma was sampled, pigs euthanized and livers collected for analyses of fatty acid contents.

Results: After 3 days of TPN the hepatic lipid deposition strongly reflected the lipid composition of TPN. Total amount of hepatic lipids was increased in S+O (33.8 mg/g) compared to the NL and S+MEN (24.6 and 26.6 mg/g respectively, $p < 0.05$). This was associated with increased amount of n-3 PUFA in the S+O (5.4 mg/g) compared with all other groups (1.2–1.5 mg/g, $p < 0.0001$). S had higher n-6 PUFA levels (9.0 mg/g) than all other groups ($p < 0.05$) and S+O had higher n-6 PUFA (7.3 mg/g) than NL and S+PEN (4–5 mg/g, $p < 0.05$). Lower levels of free fatty acids (79 μmol/L) and plasma triglycerides (0.5 mmol/L) were seen in NL compared with all other groups (110–120 umol/L, and 0.75–0.85 mmol/L, $p < 0.05$).

Conclusion: These results indicate that acute deposition of lipid in the liver closely reflects lipids profile in TPN. Further, TPN with no lipid induce the same short-term level of fat deposition in the liver as lipid-containing TPN.

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NUTRITIONAL INTERVENTION AND ORAL-SUPPLEMENT TOLERANCE IN PATIENTS WITH INFLAMMATORY BOWEL DISEASE IN ACUTE OUTBREAK EPISODE

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Rationale: Patients with IBD have high risk of malnutrition during the acute outbreak of the disease. Nutritional intervention is required in order to prevent malnutrition and its negative impact on the disease's course. Enteral nutrition (EN) is the nutritional treatment of choice

Methods: Patients by an outbreak of IBD. Nutritional Requirements; Energy (30/kg/day) protein (1.2 g/kg/day). Nutritional assessment according to nutritional risk index, albumin, % current weight loss (%CW), % normal weight (%UW) body mass index (BMI). Inflammatory markers: CRP/ESR. Oral feeding with complete formula (hypercalorical and hyperproteic) without fiber nor lactose began the first day of admission along with additional liquid diet (vegetal broth and tea), following a progressive pattern in order to achieve the nutritional requirements (30% req.first day, second day 65% and 100% third day). After the referral of acute outbreak, oral enteral nutrition was retired progressively.

Results: 31 patients (16 u.colitis, 15 Crohn), 20 women, age = 40±14.1, mean stay = 13±7 days. CRP = 5.27±5.42; ESR: 55.55±27.26. Malnutrition at admission and discharge according to albumin (71% vs 55%), by IRN at admission and discharge (87%/71%) after nutritional intervention; of albumin [3.25±0.57 admissi (p = 0.004)], IRN [89.96±11 ac discharge (p = 0.005)]. Total protein [6.26±0.69 admission vs 6.66±0.80 discharge (p = 0.000)]. Supplementation adherence 71% of patients ⩾75% met the guideline. Tolerance: 77% no nausea, no vomiting, 87% and 64% no bloating.

Conclusion: After nutritional intervention with complete polymeric hypercalorical and hyperproteicformulas without fiber nor lactose and progressive diet, we observed a significant improvement in nutritional status according to IRN, albumin and total protein.

Disclosure of Interest: None declared

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MIDDLE CHAIN TRIGLICERIDE CONTAINING ENTERAL NUTRITION SUPPLEMENTS INCREASE SERUM ALBUMIN AND NUTRITIONAL STATUS OF PATIENTS WITH CIRRHOSIS

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Rationale: Liver cirrhosis is a state of catabolism and malnutrition. Serum albumin level is not only a nutritional parameter but also a prognostic factor for cirrhosis. Middle chain triglycerides (MCT) are easily absorbed from intestine and more efficiently used by cirrhotic livers. We aimed to determine the effect of MCT-containing oral supplements on the clinical and laboratory parameters of cirrhotic patients.

Methods: 38 hospitalized cirrhotic patients (22males, median age 54) with viral etiology (29 hepatitis B, 9 hepatitis C) were retrospectively analyzed. All patients received MCT containing (50%) of oral supplement (15% egg protein, 66% carbohydrate, 19% fat, 1 kcal/ml) 480 grams/day. Child-Pugh Turcot (CPT) scores, serum prealbumin levels, NRS-2002 scores were monitored.

Results: Baseline NRS-2002 score was (median±SD) 5±1. Mean duration of nutritional supplementation was 20±3 days. Patient compliance to the treatment was 75% (95% CI: 65–90). Major reasons for discontinuation of treatment were anorexia (45%), nausea (33%), and diarrhea (12%). Mean serum albumin and prealbumin levels were (baseline and post-treatment) 2.7±1.1 vs 3.4±1.4 ($p < 0.05$) and 14±3 vs 18±4 ($p < 0.05$). Mean CPT scores were (baseline and after) 11±2 vs 8±3 ($p = 0.06$).

Conclusion: Nutritional supplementations with MCT-containing enteral solutions are effective and tolerable in patients with liver cirrhosis. Serum albumin and prealbumin levels significantly increase after treatment. However CPT scores, although indicating a favorable trend, fails to improve. Preliminary results with MCT deserve further studies probably for a longer duration in patients with liver cirrhosis.

Disclosure of Interest: None declared